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ABSTRACT

This document focuses on adolescent depression, described as a major, pervasive, and perhaps increasing problem. A brief introduction to depression in adolescence, with case examples, is followed by a discussion of what constitutes adolescent depression. Depressed mood, depressive syndromes, and clinical depression are three approaches taken in the field. An integration of these three approaches to adolescent depression is presented. A discussion of who becomes depressed in adolescence looks again at the three approaches to depression and examines variations by gender, group, and cohort. Extensive tables are provided which compare studies assessing depressed affect and studies assessing symptoms related to depression. Related disorders and problems are considered. A section on developmental processes explores both risk and protective factors, concentrating on the areas of biological processes, psychological factors, cognitive factors, family factors, peers, school factors, daily stress and stressful life events, and developmental trajectories. A section on approaches to intervention focuses on the treatment of adolescent depression, prevention of adolescent depression, and an integration of treatment and prevention efforts. A final section looks at implications for research, program, and national policy. Contains approximately 125 references and 7 tables. (NB)

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CARNEGIE COUNCIL ON
ADOLESCENT DEVELOPMENT

WORKING PAPERS

**DEPRESSION IN ADOLESCENCE:
CURRENT KNOWLEDGE, RESEARCH DIRECTIONS,
AND IMPLICATIONS FOR PROGRAMS AND POLICY**

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and Jeanne Brooks-Gunn

December 1992

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EXECUTIVE SUMMARY

Depression is a recently acknowledged problem of youth. It represents a significant concern for researchers, health professionals, and policy makers involved with adolescent development. Sad or depressed emotions are the single most salient symptom in distinguishing between youth who are referred for mental health services and those who are not referred. Therefore, the presence of depressed emotion appears to reflect significant distress and impairment in the functioning of adolescents. In addition to reflecting current distress, depressive symptoms and disorders during adolescence are predictive of depression during adulthood. Early onset of depressive disorders, including those that first occur in adolescence, have a more severe and negative course than those disorders that first occur during adulthood, further underscoring the long-term significance of depression during adolescence. Depression during adolescence is related to a host of other problem behaviors and disorders, including suicide, delinquency, conduct disorder, and substance abuse. The interrelations among these problems are complex, and depression may be involved in other disorders in a variety of ways. The tendency for depression to occur with other problems indicates that depressive symptoms may be important in understanding a variety of adolescent mental health concerns and behavioral problems.

The number of studies on adolescent depression has increased substantially in recent years. A computer search of journal articles from 1987 through 1991 using the keywords "adolescent" and "depression" yielded more than 2000 abstracts. Thus it is a propitious time to integrate the current body of knowledge.

Three related but conceptually and methodologically distinct approaches to depression are considered: (1) depressed affect or mood, (2) a syndrome of depression represented by multiple symptoms, and (3) clinical depression based on diagnosis. Depressed mood is marked primarily by negative emotions, including sadness and anxiety. An anxious/depressed syndrome has now been reliably identified from parent, teacher, and adolescent reports. A diagnosis of depression requires clinical evidence of severity, duration, and multiple symptoms. Although there is considerable similarity between depressed mood and the anxious/depressed syndrome, clinical diagnoses of depression differ in that they include somatic problems and involve long-lasting and more severe symptoms. Nevertheless, these approaches are overlapping, with significant depressed mood occurring in about one third of adolescents, approximately 15% reporting a depressive symptom, and 4% to 7% with clinical depression.

Depressed mood, depressive symptoms or syndromes, and clinical depression all appear to increase over the adolescent decade, reaching and, in some cases, exceeding the levels reported for adults. It is especially noteworthy that the higher rates of these depressive phenomena seen in adult women appear to emerge by the midpoint of the adolescent decade. Further, there is some evidence that the incidence of adolescent depression has increased since World War II.

Considerable research has now provided a picture of the factors that increase the risk for depression during adolescence. Having depressed parents is the most consistent risk factor for adolescent depression. Although growing up with a depressed parent presents an enormous risk for the development of depression and other disorders in

offspring, not all children of depressed parents display dysfunctional patterns of development. Therefore, comparison between siblings within families of depressed parents is an important avenue for future research. Comparisons among siblings whose parents are depressed may offer clues to the non-shared characteristics of family environments and genetic factors which either increase the risk for disorder or protect children from disorder in these families. Further, such studies should be useful in constructing preventive interventions for families of depressed parents, as well as provide information about risk and protective factors for adolescent depression.

Depressive affect, symptoms, and disorders also occur in adolescents whose parents are not depressed suggesting that additional risk factors are involved. Being adolescent and female both appear to be strong predictors for most aspects of depression. Stressful life events and minor chronic stresses and strains are linked to depression and depressed affect. There is some evidence linking other factors to depression in a presumably causal way but research in most areas is at an early stage. There has been much less research in protective factors except for two areas. Good parenting appears to protect from adolescent depression and positive peer relationships seem to be protective beginning in mid-adolescence.

The development of depression has been little examined, but the etiology of adolescent depression is likely to be similar to the etiology of depression at other ages. Twin studies suggest some genetic components that may confer increased vulnerability to depression or perhaps vulnerability to the biological dysregulation that accompanies episodes of depression. Periods of depression that require hospitalization or reduced

social activity and contact with peers are likely to be detrimental to the developing adolescent, producing increased morbidity and perhaps comorbidity with other disorders. Briefer periods of depressed feelings may be quite normative and involve total recovery, at least starting in middle adolescence. Whether the individual knows how to cope effectively with stress and depressed feelings appears to be a major factor in the subsequent developmental trajectory over adolescence and into adulthood.

An understanding of the developmental course of depression during adolescence cannot be limited to studies of the adolescent period. Several longitudinal data sets have now been acquired on depressive symptoms and disorders during childhood. It will be important to follow the development of these children as they enter adolescence to identify potential early markers of adolescent depression. Should a set of early psychological, social, and biological markers be identified, they could provide a strong basis for early preventive interventions.

More severe depression is likely to be accompanied by other problems, termed comorbid disorders. These include other internalizing disorders (e.g., anxiety disorders), as well as externalizing disorders (e.g., conduct disorders). In addition, some adolescents may cope with depression in ways that result in other problems, such as drinking or drug use. Such covariance of problems is likely to indicate more serious difficulties. Suicide appears to have an independent etiology from depression, although suicidal thoughts and behaviors frequently co-occur with depression.

Psychosocial treatments appear to have some effectiveness with adolescent depression. Controlled studies of pharmacotherapy suggest that this treatment alone

does not differ significantly from a placebo control treatment. The reasons that pharmacologic treatments with adolescents are less effective than with depressed adults are not known. The mechanisms underlying positive responses to psychosocial and pharmacologic interventions are also not known, either at the psychological or biological level. Whether the two approaches have differential effectiveness for particular symptoms or subgroups of depressed adolescents is also unknown. Therefore, further research is needed to identify the specific components of interventions that are responsible for the remission of depressive symptoms.

Preventive interventions are just beginning to be tested, but are promising for providing adolescents with effective coping skills that might reduce the incidence of depressive phenomena. Given the cumulative nature of the developmental effects of poor coping, efforts to prevent negative developmental trajectories seem worthwhile. Further, given the high risk for depression among adolescents whose parents are depressed, preventive interventions for these youth should be a high priority.

Several implications for policy result from this synthesis of research. As for research policy, the many important findings within biological and psychosocial areas point to the need for studies that integrate these areas. Although we now have sufficient data to recognize that depression represents a significant problem during adolescence, the mechanisms that explain *how* and *why* depression develops and, in some cases persists, during adolescence are less well understood. Studies are also needed that differentiate developmental patterns and correlates of major depressive disorder versus depressed mood.

A major program implication is the need to intervene with children of depressed parents, preferably as a family. Adolescent service programs should recognize the extensive overlap of depression with other disorders and problems and provide integrated services. Similarly, federal and state policies should permit an integrative focus on the adolescent rather than on a specific problem.

Adolescent depression is a major, pervasive, and perhaps increasing problem. A sufficient knowledge base exists now to guide action in research, prevention, and treatment. Significant morbidity of adolescence could be ameliorated by attention to adolescent depression.

Depression in Adolescence

Thirteen-year-old Tony was moody and irritable at home. He didn't seem to want to discuss anything with his parents and he didn't want to do anything his parents suggested. He did spend time on the telephone with his friends. He went to school every day, but didn't complete his homework.

Fifteen-year-old Carla seemed sad to her parents and her teachers, and disinterested in everything, even things she used to enjoy. Her teachers had commented that she seemed unable to concentrate in school and always seemed to be very tired. This concerned her parents because at home she seemed only to sleep, with little interest even in eating. Although she had periods like this before, they had never lasted very long. This time these symptoms had lasted three weeks.

Since seventeen-year-old John had broken up with his girlfriend, he seemed to have lost interest in everything. After the breakup, John found himself crying when alone and felt generally worthless. At a party where he saw his former girlfriend with another guy, John then drank heavily. While driving home, he found himself thinking about taking a sharp corner at very high speed.

All of these scenarios describe situations that would probably be identified with adolescence even if the ages of these young people were omitted. Should the behavior

be of little concern and dismissed as simply "adolescent behavior?" Or should such incidents worry parents, teachers, or friends? All three situations would surely cause some concern, but they probably represent different aspects of depression based on the limited information provided. Although clearly manifesting depressed affect, Tony would not meet the criteria for a clinical diagnosis of depression. Carla, on the other hand, is most likely clinically depressed. John is upset about the breakup with his girlfriend and thinking about suicide. Although suicidal thoughts are a matter of concern in youth, they may or may not be accompanied by depression.

What is Adolescent Depression?

The image of adolescence as a time of storm and stress, intense moodiness, and preoccupation with the self has permeated both professional and lay perspectives on this developmental period. The belief that significant difficulties during adolescence represent normal healthy development has had two major effects on research and practice: (1) difficulties during adolescence were not considered important, and (2) adolescent problems were often not treated because of the belief that the adolescent would grow out of them.

Although this view of adolescence is the one commonly reflected in the media and many clinical descriptions of adolescence, it is not supported by research on this period. In the late 1960s, several reports documented adolescents who traversed this period of life without significant psychological difficulties (Douvan & Adelson, 1966; Offer, 1969). It is now known that the majority of adolescents of both genders successfully negotiate

this developmental period without any major psychological or emotional disorder, develop a positive sense of personal identity, and manage to forge adaptive peer relationships at the same time that they maintain close relationships with their families (Powers, Hauser, & Kilner, 1989). Research in the 1970s on youth with problems demonstrated that psychological difficulties in adolescence frequently developed into serious psychiatric disorders in adulthood (Rutter, Graham, Chadwick, & Yule, 1976; Weiner & DelGaudio, 1976). These and other studies demonstrated the inaccuracy of the belief that difficulties such as depression were normal manifestations of adolescence, and pointed toward the need for assessment, diagnosis, prevention, and treatment at this age. These studies also highlighted the need for more research on the development of depression in adolescence.

The dramatic change in knowledge about adolescence has had clear effects on recent research. Now that depression, depressive symptoms, and depressed mood have been recognized as appropriate subjects of investigation in adolescents, the number of studies on these topics has exploded. From our computerized literature search on the key words "adolescent" and "depression" for entries published from 1987 through 1991, more than 2000 entries were identified. There now is a critical mass of knowledge that can benefit from a synthesis such as the present one.

Three approaches to the assessment and classification of adolescent psychopathology have been reflected in the literature on adolescent depression: (1) depressed mood, (2) depressive syndromes, and (3) clinical depression. Each approach reflects different assumptions about the nature of psychopathology, serves

different purposes, and reflects different aspects of depression. For example, the study of depressed mood during adolescence has emerged from developmental research in which depressive emotions are studied along with other features of adolescent development. The depressive syndrome approach assumes that depression and other syndromes reflect the co-occurrence of behaviors and emotions that deviate from the typical. The clinical approach is based on assumptions of a disease or disorder model of psychopathology.

Depressed Mood

Like Tony in the example above, everyone experiences periods of sadness or unhappy mood at various points in their lives. These periods of depressed mood may occur in response to many situations, such as the loss of an important relationship or failure on an important task. They may last for a brief or long period of time. And they may be associated with no other problems, or many problems. Depressed mood is usually experienced with other negative emotions, such as fear, guilt, anger, contempt, or disgust (Watson & Kendall, 1989). Depressed mood during adolescence is frequently present when any of these other negative emotions are present (Saylor, Finch, Spirito, & Bennett, 1984). Depressed mood is also likely to be linked with other problems, such as anxiety or social withdrawal. Although anxiety and depressed mood frequently occur at the same time, anxiety may or may not be related to positive moods; in contrast, depressed mood is not present when one feels happy, or conversely, happy mood does not occur at the same time as depressed mood (Watson & Kendall, 1989). The presence of depressed mood has been found to be the single most powerful symptom

differentiating those referred or not for clinical treatment (Achenbach, 1991b,d).

Depressive Syndromes

In the earlier vignette, John had feelings of worthlessness, loneliness, and crying, with suicidal thoughts. He would probably score high on a depressive syndrome, as well as self-destruction. Approaches that look at many symptoms and problems, including depression, show that aspects of depression are found in many other problems (Achenbach, 1991a). This approach considers a syndrome of complaints that include both anxiety and depression, and is based on items such as lonely, cries, fears doing bad things, needs to be perfect, unloved, feels others are out to get him/her, worthless, nervous, fearful, guilty, self-conscious, suspicious, sad, and worries (Achenbach, 1991a,b,c). Scores on this syndrome are very strongly related to seven other problem syndromes identified by this approach: Withdrawn, Somatic Complaints, Social Problems, Thought Problems, Attention Problems, Delinquent Behavior, Self-Destructive, and Aggressive Behavior. Table 1 shows these relationships for the Youth Self-Report (YSR) and the Child Behavior Checklist (CBCL), completed by parents. The weakest relationship occurred with the Delinquent Behavior syndrome, and the strongest relationships were found with the Self-Destructive and Withdrawn syndromes. John would be considered as having self-destructive tendencies with significant negative mood, but would probably not be considered clinically depressed.

Clinical Depression

Carla, the second adolescent described in initial vignettes would probably be diagnosed with clinical depression. There are two major diagnostic models typically used

Table 1

Correlation of Anxious/Depressed Syndrome With Other Syndromes on the Youth Self Report and Child Behavior Checklist

Syndrome	Youth Self-Report			
	Boys		Girls	
	Non-Referred	Clinically Referred	Non-Referred	Clinically Referred
Withdrawn	.62	.61	.62	.61
Somatic Complaints	.47	.49	.57	.51
Social Problems	.45	.54	.47	.52
Thought Problems	.35	.46	.50	.40
Attention Problems	.61	.66	.63	.60
Delinquent	.39	.27	.45	.31
Aggressive	.49	.49	.56	.51
Self-Destructive	.75	.80	---	---
Mean	.52	.54	.54	.49

Syndrome	Child Behavior Checklist			
	Boys		Girls	
	Non-Referred	Clinically Referred	Non-Referred	Clinically Referred
Withdrawn	.58	.61	.65	.65
Somatic Complaints	.28	.45	.43	.48
Social Problems	.50	.42	.51	.41
Thought Problems	.49	.53	.54	.54
Attention Problems	.60	.54	.55	.54
Delinquent	.41	.25	.44	.30
Aggressive	.59	.47	.63	.51
Mean	.49	.47	.54	.49

Note. From Achenbach, 1991a.

to diagnose clinical depression: the categorization of mental disorders developed by the American Psychiatric Association (1987) and the method developed by the World Health Organization (1990). The American Psychiatric Association method is the one most widely used in the United States and abroad (Maser, Kaelber, & Weise, 1991). It bases the diagnosis of disorders on a review of the presence, duration, and severity of sets of symptoms. Under depressive disorders, adolescents may be diagnosed as experiencing Major Depressive Disorder (MDD) and/or Dysthymic Disorder.

To meet the criteria for Major Depressive Disorder, the adolescent must have experienced five or more of the following symptoms for at least a two week period at a level which differs from prior functioning: (1) depressed mood or irritable mood most of the day, (2) decreased interest in pleasurable activities, (3) changes in weight or perhaps failure to make necessary weight gains in adolescence, (4) sleep problems, (5) psychomotor agitation or retardation, (6) fatigue or loss of energy, (7) feelings of worthlessness or abnormal amounts of guilt, (8) reduced concentration and decision-making ability, and/or (9) repeated suicidal ideation, attempts or plans of suicide. The diagnosis of Major Depressive Disorder in adolescents is further differentiated by severity and chronicity of the symptoms. A range of mild, moderate, and severe diagnoses with or without psychotic features is applied. For example, many depressive symptoms that significantly affect the functioning of the individual in school, work, or other social domains would be classified as severe. Psychotic features would be behaviors such as delusions or hallucinations. Classification as a chronic disorder requires that the depression last

over two consecutive years in which there have not been more than two months without symptoms.

A Dysthymic Disorder is diagnosed when the adolescent has had a period of at least one year in which the individual shows depressed or irritable mood every day without more than two months symptom-free. In addition, Dysthymic Disorder requires the presence of at least two of the following symptoms: (1) eating problems, (2) sleeping problems, (3) lack of energy, (4) low self-esteem, (5) reduced concentration or decision-making ability, and/or (6) feelings of hopelessness. There cannot be an episode of Major Depressive Disorder during the first year of Dysthymic Disorder. Primary Dysthymia is unrelated to nonmood disorders such as Eating Disorders, Substance Abuse Disorders, or Anxiety Disorders; any of these would classify the disorder as a secondary Dysthymic Disorder. Occurrence of Dysthymic Disorder prior to 21 years of age is considered early onset. An episode of Major Depressive Disorder following the onset of Dysthymic Disorder is typically referred to as "double depression."

Integration of Approaches

To compare the core features of the three approaches, we have summarized in Table 2 items measuring each from sample assessments. For depressed mood, we list items from the Emotional Tone Scale (Offer, Ostrov, & Howard, 1982; Petersen, Schulenberg, Abramowitz, Offer, & Jarcho, 1984) and the Child Depression Inventory (CDI) Negative Affect with Somatic Symptoms factor (Weiss, Weisz, Politano, Carey, Nelson, & Finch, 1991). The anxious/depressed syndrome is represented by items from the measures developed by Achenbach (1991a): Child Behavior Checklist (CBCL), Youth

Self Report (YSR), and Teacher Report Form (TRF). Clinical depression is represented by the symptoms of Major Depressive Disorder (MDD) from DSM-III-R.

As can be seen in Table 2, there is considerable overlap in the depressed mood and depressive syndrome approaches to adolescent depression; note, however, that only depressed mood and low self-esteem or feelings of worthlessness link the first two approaches with a clinical diagnosis of depression. The differences among the three approaches involve anxiety and somatic symptoms. Anxiety is involved with both depressed mood and the depressive syndrome but not clinical depression. Conversely, a clinical diagnosis of depression also requires somatic problems such as sleep and appetite problems, psychomotor problems, and fatigue; these symptoms may or may not be present with the other two approaches.

Empirical studies suggest significant overlap in the approaches. Clinically depressed adolescents score high on the depression syndrome (Edelbrock & Costello, 1988; Rey & Morris-Yates, 1991; Weinstein, Noam, Grimes, Stone, & Schwab-Stone, 1990). Similarly scores on the depressive syndrome are highly related to depressed mood (Petersen, 1990, March). The fact that depressed mood and the depressive syndrome are usually based on questionnaires, whereas a diagnosis is usually obtained in an interview may influence similarities or differences in the reports. Considered together, the data suggest that there is a continuum running from depressed mood through depressive syndrome to depressive disorder. Negative affect is common to all three approaches (Compas, Ey, & Grant, 1992), but the chronicity of negative affect is one dimension that differentiates the three approaches, with clinical diagnosis being the

Table 2

Comparison of Items from Core Syndrome Anxious/Depressed on the Child Behavior Checklist (CBCL), Youth Self Report (YSR), and Teacher Report Form (TRF), Emotional Tone Scale, DSM-III-R Criteria for Major Depressive Disorder (MDD) and the Child Depression Inventory (CDI).

CBCL, YSR, and TRF for Anxious/Depressed Syndrome	Emotional Tone Scale	MDD from DSM-III-R	CDI
*Unhappy, sad, depressed *Feels worthless *Lonely *Feels too guilty *Cries a lot Fears impulses Needs to be perfect Feels unloved Feels persecuted Fearful, anxious Self-conscious Suspicious Worries	*I frequently feel sad *Feel I am not as good as most people *I feel so very lonely I am not a person I would like to be I feel relaxed	*Depressed mood *Feelings of worthlessness *Feelings of guilt	*I am sad all the time *I feel like crying every day Things bother me all the time

Note. *Items common to at least two of the four approaches.

Table 2 (continued)

CBCL, YSR, and TRF for Anxious/Depressed Syndrome	Emotional Tone Scale	MDD from DSM-III-R	CDI
<u>Specific to YSR</u> *Thinks about suicide Harms self <u>Specific to TRF</u> Overconforms *Hurt when criticized Anxious to please Afraid to make mistakes	 *Feelings are easily hurt Feel emotionally upset *I enjoy life	*Recurrent thoughts about death, suicidal ideation *Diminished interest or pleasure *Weight loss or gain *Insomnia or hypersomnia Psychomotor agitation or retardation *Fatigue or loss of energy Diminished ability to think or concentrate	 I worry about aches and pains *I do not feel like eating *I have trouble sleeping *I am tired all the time Hopeless

Note. *Items common to at least two of the four approaches.

most specific and extensive. None of the three approaches include biological factors as criteria; this is not surprising given the recency of biological methods for studying depression and the lack of evidence for their validity at present.

It also appears that depressed mood cannot be distinguishable from anxious mood in adolescents (Finch, Lipovsky, & Casat, 1989). Similarly, the depressive syndrome includes anxiety as well (Achenbach, 1991a). A clinical diagnosis of depression ignores anxiety except that its presence with dysthymia would yield a diagnosis of Secondary Dysthymic Disorder.

Who Becomes Depressed in Adolescence?

There are no nationally representative studies of depression in adolescents. Especially lacking is information on the incidence of depression in various ethnic groups and social classes. At the same time, there are increasing numbers of studies using the three approaches just described to provide some information on rates of depressive mood, syndromes, and disorders in some samples of adolescents.

Depressed Mood

Most of the studies conducted more recently assess what we have called depressed mood. (See Table 3). There has especially been a rapid increase in studies in the past two years. Although these studies included different measures with somewhat different items and sampled symptoms over varying periods of time, some tentative conclusions can be drawn from these data. The thirty studies we were able to identify are based entirely on non-clinical samples. Although studies of depressed mood typically examine the phenomenon in relation to other problems or aspects of adolescent

Table 3

Studies Assessing Depressed Affect

	Albert & Beck (1975)	Leon et al. (1980)	Frerichs, et al. (1981)	Kandel & Davies (1982)	Schoenbach et al. (1982)	Teri (1982)	Kaplan et al. (1984)	Tolor & Murphy (1985)	S. Cohen et al. (1986)	L. Cohen et al. (1987)
Number of subjects	63	138	1003	4202	384	568	385	325	609	312
Age of subjects	11-15	13-16	18-24	14-18	12-15	14-17	11-18	13-17	15-27 (M=19 yr)	7th-8th graders (M=13 yr)
Informants ¹	A	A,P,T	A	A,P	A	A	A	A	A	A,P
Study Duration	2 wks	2 wks	current	1 yr	1 wk	2 wks	2 wks	6 mths	6 mths	1 yr
Instrument ²	Short BDI quest.	quest. (incl. PIC & CDI)	quest.	6 item quest.	quest. (incl. CES-D)	quest. (incl. BDI)	quest. (incl. BDI)	quest. (incl. CES-D)	quest. (incl. CES-D)	quest. (incl. CDI) & interviews
Percentage above "diagnosis cut-off" ³	39	15	-	18,28 ⁴	3,50 ⁵	49	22	-	-	-
Gender effects ⁶	-	-	G>B	G>B	-	-	no	G>B	-	-
Age effects ⁷	-	-	-	no	-	-	LA>MA	-	-	-

1. A=Adolescent; P=Parent; T=Teacher.

2. BDI=Beck Depression Inventory; CDI=Children's Depression Inventory; CES-D=Center for Epidemiologic Studies Depression Scale; PIC=Personality Inventory for Children.

3. >9 for the BDI; >16 for the CES-D; >12 for the CDI.

4. 18% is the rate relative to the mean of the scale with a clinical sample, 28% is the rate relative to a different cutoff identifying extreme depression.

5. 3% is the rate using RDC criteria, 50% using CES-D cutoff.

6. G=girls; B=boys.

7. MA=middle adolescence; LA=late adolescence.

Table 3 continued

	Fauber et al. (1987)	Siegel & Brown (1988)	Rierdan et al. (1988)	Friedrich et al. (1988)	Gjerde, et al. (1988)	Reinherz et al. (1989)	Ostrov, et al. (1989)	Petersen et al. (1990)	Vernberg (1990)	Garrison et al. (1990)
Number of subjects	89	364	103	269	106	378	497	335	73	550
Age of subjects	11-15	10-17	9th graders (M=15 yr)	13-16	18	15	16-18	11-21 longitudinal	7th-8th graders	11-15
Informants ¹	A,P,T	A	A	A,M	A	A,M	A	A,P	A	A
Study Duration	1 yr	1 yr	current	current	15 yrs	10 yrs	current	10 yrs	6 mths	3 yrs
Instrument ²	quest. (incl. CES-D)	quest. (incl. CES-D)	quest. (incl. BDI)	quest. (incl. BDI)	quest. (incl. CES-D)	quest. (incl. CDI) & inter.	quest. (incl. OSIQ, DCL, SCL)	quest. (incl. ET) & inter.	quest. (incl. CDI)	quest. (incl. CES-D)
Percentage above "diagnosis cut-off" ³	-	-	47	37	-	21	-	50 ⁴	-	-
Gender effects ⁵	-	-	G>B	G>B	no	G>B	G>B	G>B	-	G>B
Age effects ⁶	no	-	no	-	-	no	-	ET: EA>MA>LA DE: MA>EA,LA	-	-

1. A=Adolescent; M=Mother; P=Parent; T=Teacher.

2. BDI=Beck Depression Inventory; CDI=Children's Depressive Inventory; CES-D=Center for Epidemiologic Studies Depression Scale; DCL=Delinquency Checklist; ET=Emotional Tone Scale of the Self-Image Questionnaire for Young Adolescents; OSIQ=Offer Self-Image Questionnaire; SCL=Symptom Checklist.

3. >9 for the BDI; >16 for the CES-D; >12 for the CDI.

4. In 12th grade, 50% reported having experienced a depressive episode (DE) lasting two weeks or more since the beginning of high school.

5. G=girls; B=boys.

6. EA=early adolescence; MA=middle adolescence; LA=late adolescence.

Table 3 continued

	Larsson & Melin (1990)	Allgood et al. (1990)	Paikoff et al. (1991)	Kandel, et al. (1991)	Seiffge- Krenke (1991)	Richter & Fend (1991)	Susman et al. (1991)	Bernstein (1991)	Garber & Braafladt (1991)	Roberts et al. (1991)
Number of subjects	547	802	103 (girls only)	597	136	1399	108	96	275	1704
Age of subjects	13-18	13-18	10-14	9th & 11th graders	14-18	16-18	9-15	7-17	k-8th graders	9th-12th graders
Informants ¹	A	A	A	A	A	A	A	A	A,M	A
Study Duration	5 mths	1 mth	1 yr	current	current	current	2 yrs	current	current	1 mth
Instrument ²	quest. (incl. BDI)	OSIQ, CES-D	CBCL, CES-D	quest. (incl. Kandel Scale)	quest. (incl. YSR)	quest. (incl. BDI)	quest. (incl. OSIQ, CBCL, DISC)	quest. (incl. CDRS-R, CDI,CDS)	quest. (incl. CDI,CAQ)	quest. (incl. BDI,CES-D)& psychiat. interviews
Percentage above "diagnosis cut-off" ³	-	-	-	-	-	33	-	53	21	28,48 ⁴
Gender effects ⁵	G>B	G>B	-	-	-	no	-	-	-	-
Age effects	no	no	-	-	-	-	-	-	-	-

1. M=mother; A=adolescent.

2. BDI=Beck Depression Inventory; CAQ=Child Affect Questionnaire; CBCL=Child Behavior Checklist; CDI=Children's Depression Inventory; CDRS-R=Children's Depression Rating Scale (Revised Form); CDS=Children's Depression Scale; CES-D=Center for Epidemiologic Studies Depression Scale; DISC=Diagnostic Interview Schedule for Children; OSIQ=Offer Self-Image Questionnaire; YSR=Youth Self-Report.

3. >9 for the BDI; >16 for the CES-D; >12 for the CDI.

4. 28% were depressed using the cutoffs of the BDI; 48% were depressed using the cutoff of the CES-D.

5. G=girls; B=boys.

development, some investigators of depressed mood have identified a threshold above which a score is thought to be predictive of clinical depression. Using such scores, the median rate of significantly elevated depressed mood in fourteen studies is 35 percent. (When two cutoffs were identified in a single study, the average [mean] of the two was used to calculate the median. The seventh score was 33%; the eighth was 37%). Not unexpectedly, these represent much higher rates than the average calculated from studies examining clinical depression.

All but three of the fourteen studies examining gender effects find differences, in all cases with girls reporting more depressed affect than boys. Of ten studies examining age effects, most find no such effects. However, only two of these studies were actually longitudinal, following the same subjects over time. In two longitudinal studies, depressed affect appears to decrease with age for boys, but remains level across time for girls (Block, 1991; Petersen, White, & Stemmler, 1991, April). A different pattern of developmental change has also been reported: Radloff (1991) found dramatic increases in depressed moods between ages 13 and 15 years, a peak around 17-18 years, with a subsequent decline to adult levels. This pattern has also been found with a measure of depressive episodes in which the mid-adolescence peak is higher for girls than boys (Petersen et al., 1991, April). Elevated rates of depressed mood in adolescence relative to adulthood have been reported in other studies as well (e.g., Allgood-Merten, Lewinsohn, & Hops, 1990; Larsson & Melin, 1990).

Depressive Symptoms and Syndrome

Studies assessing symptoms related to depression are summarized in Table 4. In eleven studies, the average percentage reporting sad or miserable symptoms is 15%. (Many of the studies in the table included children as well as adolescents. The figure we estimate is based on adolescents in those studies where information was provided separately for this age group.) The table demonstrates remarkable variation among the percentages for any specific symptom over the many studies. Characteristics of the samples are generally not reported.

Research examining depressive syndromes has used a cut-off score corresponding to the 95th percentile in a nationally representative sample of adolescents to identify a clinical range (Achenbach, 1991a,b,c). This score was identified as having the optimal power for discriminating between clinically-referred and non-referred samples with the lowest rates of false positives and false negatives. Thus, this approach to measuring depressive syndromes has established an empirically-based rate of 5% of the normal population in the clinical range on a depressive syndrome at any given time.

Clinical Depression

Table 5 summarizes the 21 studies that report a clinical diagnosis of depression. The percent of adolescents receiving a diagnosis of depression varies from near zero in a large non-clinical sample of children aged 10 to 11 years (Rutter, Tizard, & Whitmore, 1970) to 57% in a clinical sample from 8 to 13 years (Kovacs, Feinberg, Crouse-Novak, Paulauskas, & Finkelstein, 1984a). Studies based on clinical samples, of course, tend to yield higher depression rates (averaging 42%, median is 48%, across six studies). The

fourteen studies of non-clinical samples yield an average of 7% depressed, with a median of 4%. (The studies of children of depressed parents were not included in the averages because these are samples at high risk for depression. Where the samples include the rate for a control group, that figure is included. For the reader's information, we include the data in the table).

Summary

Epidemiological studies suggest that clinical depression is a serious problem in adolescence, affecting three to seven percent of young people during the adolescent decade. Of those being treated for a clinical problem in adolescence, about 42% to 48% have the primary diagnosis of depression. The depressive symptom of sadness is reported by about 15 percent of adolescents. Depressed mood, however, is even more pervasive; about one-third of adolescents report depressed mood, with girls more affected than boys. Note that the rates increase with each approach from clinical depression to depressive symptoms, and from depressive symptoms to depressed mood.

Clinical depression as well as depressed mood appear to increase dramatically in adolescence compared to childhood (Rutter et al., 1976; Rutter, 1986). Preadolescent onset of clinical depression is considered to be a serious risk factor for adult depression or other major mental disorders (Harrington, Fudge, Rutter, & Pickles, 1990; Kovacs et al., 1984a,b). The appearance of clinical depression in adolescence also predicts increased likelihood for adult depression or other major mental disorders in adulthood. Rates of clinical depression are not higher in adolescence than adulthood but rates of depressed mood do appear to be higher in adolescence, based both on longitudinal and cross-sectional studies.

Table 4¹

Studies Assessing Symptoms Related to Depression

	Mc Fie (1934)	Griffiths (1952)	MacFarlane et al. (1954)	Lapouse & Monk (1964)	Cullen & Boundy (1966)	Rutter et al. (1970)	Shepard et al. (1971)
Number of subjects	697	3387 parents 900 children	126	482	3440	2199	7004
Age of subjects	12-14	6-14	2½-14 ³	6-12 ³	2-15	10-11	5-15 ³
Informants ²	T	T/P/C	P	P	P	P,T,C	T,P
PERCENTAGE REPORTING SYMPTOMS							
Sad/ miserable:	1	-	10-30 ⁴	-	1	12	-
Crying:	-	-	-	-	-	-	5
Moody:	-	-	-	-	2	-	15
Withdrawn:	-	-	-	-	1	-	-
Low appetite:	-	-	-	20	-	21	5
Increased appetite:	-	-	<1	16	-	-	20
Poor sleep:	-	-	20	-	-	19	10
Increased sleep:	-	-	-	-	-	-	-
Constipation:	-	-	-	-	-	-	5
Underactive:	-	-	<5	-	-	-	6
Feels guilty:	-	-	-	-	-	-	-
Feels worthless:	-	-	-	-	-	-	-
Feels unloved:	-	-	-	-	-	-	-
Irritable:	-	-	10	-	-	21	11
Stomach pains:	-	-	-	-	-	33	2
Headaches:	-	-	-	-	-	10	6
Whining:	-	-	-	-	-	-	3
Fear of school:	-	-	-	8	-	-	-
Lonely:	-	-	-	-	-	-	-
Low concentration:	-	-	-	16	-	22	-
Nightmares:	-	-	<2	28	-	-	15
Overtired:	-	-	-	-	-	-	-
Nervous/anxious:	14	50	-	-	26	14	-
Worrying:	-	75	-	-	-	21	11
Suicidal:	-	-	-	-	-	8	-

1. Based on Angold (1988); percentages are rounded off.

2. T=teacher; P=parent; C=child. Where more than one informant was employed in a study, the figures given always refer to parent reports.

3. Only results related to adolescents are reported.

4. Numbers represent minimum and maximum value of the studied age range.

Table 4¹ continued

	Albert & Beck (1975)	Rutter et al. (1976)	Achenbach Edelbrock (1981) ³	Schoenbach et al. (1982)	Teri (1982)	Kaplan et al. (1984)	Verhulst et al. (1985)
Number of subjects	63	2303	1300	384	568	385	2076
Age of subjects	11-15	14-15	4-16	12-15	14-17	11-18	4-16
Informants ²	C	P,T,C	P	C	C	C	P
PERCENTAGE REPORTING SYMPTOMS							
Sad/							
miserable:	29	45	9 (0)	15	5	3	<5-10 ⁴
Crying:	-	-	10 (2)	14	11	8	0-<25
Moody:	-	-	22 (3)	-	-	-	<20-<35
Withdrawn:	35	-	6 (0)	-	4	2	<5-<15
Low appetite:	37	-	20 (4)	14	8	5	<10-<40
Increased							
appetite:	-	-	16 (4)	-	-	-	10-20
Poor sleep:	-	-	12 (4)	21	7	6	<10-<30
Increased sleep:	38	-	7 (1)	24	-	-	<10-<15
Constipation:	-	-	6 (1)	-	-	-	<5-10
Underactive:	-	-	5 (1)	-	-	-	5-<20
Feels guilty:	-	-	6 (0)	-	4	2	10-10
Feels worthless:	-	-	13 (1)	-	-	-	<5-<5
Feels unloved:	-	-	22 (1)	-	-	-	<10-<20
Irritable:	-	-	44 (5)	-	14	9	<30-<50
Stomach pains:	-	-	10	-	-	-	<20-20
Headaches:	-	-	12	-	-	-	<5-<25
Whining:	-	-	19 (2)	-	-	-	-
Fear of school:	-	-	3 (0)	-	-	-	<5
Lonely:	-	-	20 (3)	-	-	-	<5-20
Low concentration:	-	-	32 (6)	30	7	-	30-60
Nightmares:	-	-	19 (2)	-	-	5-<20	-
Overtired:	44	-	17 (2)	-	-	-	10-<20
Nervous/anxious:	30	-	22 (4)	-	-	2	20-<50
Worrying:	-	21	25 (2)	-	-	-	<5-<25
Suicidal:	33	8	1 (0)	-	5	4	0-<5

1. Based on Angold (1988); percentages are rounded off.

2. T=teacher; P=parent; C=child. Where more than one informant was employed in a study, the figures given always refer to parent reports.

3. Figures supplied by Craig Edelbrock based on data reported in Achenbach & Edelbrock (1981). Figures in parentheses refer to frequency of a severe (2) rating; the preceding figure represents the frequency of a rating of 1 or 2 on that item.

4. Numbers represent minimum and maximum value of the studied age range.

Table 5
Summary of Studies Employing a Diagnostic Nosology for Clinical Depression

	Rutter et al. (1970)	Leslie (1974)	Rutter et al. (1976)	Frost et al. (1984) ¹	Kovaks et al. (1984)	Levy et al. (1984) ²	Kashani et al. (1987)	Hammen et al. (1987)	Bird et al. (1988)	Edelbrock et al. (1988)
Number of subjects	2199	1198	2303	855	114	424	150	64	843	270
Age of subjects	10-11	13-14	14-15	3-13	8-13	16-19	14-16	8-16	4-16	6-16
Population ³	NC	NC	NC	NC	C	NC	NC	NC ⁴	NC	C
Informants ⁵	A,P,T	A,P,T	A,P,T	A	A,P	A	A,P	A,P	A,P,T	A,P
Time period	Current ⁶	Current	Current	10 yrs	5 yrs	Current	Current	6 mths	Current	Current
Instrument ⁷	Screen quest. & psychiat. interviews	Screen quest. & psychiat. interviews	Screen quest. & psychiat. interviews	DISC, RBPC, RCSB	ISC	DIS	Screen quest. & psychiat. interviews	K-SADS CBCL	CBCL, DISC & psychiat. interviews	DISC,CBCL
Percentage with diagnosis of depression	0.14	1	2	2	57	7(LP) ⁸	5	DP=45 ⁴ NDP=26	6	48

1. Refers to the Dunedin Multidisciplinary Health and Development Study. Whenever there were several articles published on the same sample, only one which represents this study is listed.

2. Refers to a sample of college students from the Boston Area. Whenever there were several articles published on the same sample, only one which represents this study is listed.

3. C=Clinical; NC=Nonclinical

4. Sample consisted of children of parents with (DP) and without (NDP) a major depressive disorder.

5. T=teacher; P=parent; M=mother; A=adolescent. Figures quoted are obtained from children self-reporting symptoms, except for Isle of Wright studies where rates by parent reports are given.

6. All children with a psychiatric disorder were assessed longitudinally.

7. CBCL=Child Behavior Checklist; CDI=Children's Depression Inventory; DISC=Diagnostic Interview Schedule for Children; ISC=Interview Schedule for Children; K-SADS=Schedule for Affective Disorders and Schizophrenia (Version for school-aged children); PIC=Personality Inventory for Children; RBPC=Revised Behavior Program Checklist; RCSB=Rutter Child Scale B.

8. LP=Lifetime prevalence.

Table 5 (continued)

	Garber et al. (1988)	Fleming et al. (1989)	Kashani et al. (1989)	Mitchell et al. (1989)	Velez et al. (1989)	Daniels & Moos (1990)	Fendrich et al. (1990)	Weinstein et al. (1990) (1991)	Grigoriou- Serbănescu et al.	Lewinsohn et al. (1991)	Rey et al. (1991)
Number of subjects	20	2852	210	132	776	190	220	160	120	1710	667
Age of subjects	M=14	6-16	8,12,17	7-17	9-18	12-18	6-23 (12-18 N=92)	11-16	10-17	14-18	12-16
Population ¹	C	NC	NC	C	NC	NC,C	NC ²	C	NC ²	NC	C
Informants ³	A	A,P,T	A,P	A,P	A,M	A	A,P	A	A	A	A,P
Time period	8 yrs	Current	Current	Current	8 yrs	Current	Current	Current	Current	Current	Current
Instrument ⁴	Screen quest. & psychiat. interviews	SDI	CAS, P-CAS	K-SADS	DISC, DISC-P	Screen quest. & psychiat. interviews	K-SADS- E	DISC, YSR	K-SADS-E	K-SADS	CBCL & psychiat. interviews
Percentage with diagnosis of depression	55	2	3	29	3	26	22 = NDP	35 = DP ² 48	0 = NDP	8 = DP ² 20	13

1. C=Clinical; NC=Nonclinical

2. Sample consisted of children of parents with (DP) and without (NDP) a major depressive disorder.

3. T=teacher; P=parent; M=mother; A=adolescent. Figures quoted are obtained from children self-reporting symptoms, except for Isle of Wright studies where rates by parent reports are given.

4. CAS=Child Assessment Schedule; CBCL=Child Behavior Checklist; CDI=Children's Depressive Inventory; DISC=Diagnostic Interview Schedule for Children; ISC=Interview Schedule for Children; K-SADS=Schedule for Affective Disorders and Schizophrenia; P-CAS=Child Assessment Schedule (Parent Version); NC=Personality Inventory for Children; RBBP=Revised Behavior Problem Checklist; RCB=Revised Child Scale; YSR=Youth Self-Report

Variations by Gender, Group, and Cohort

Gender differences. All the evidence suggests that increases in depressive disorders and mood are greater for girls than for boys during adolescence (Kandel & Davies, 1982; Kashani et al., 1987; Petersen, Kennedy, & Sullivan, 1991a). The gender difference that emerges by age 14 to 15 years appears to persist into adulthood. Many scholars have considered whether the gender difference is a true difference in depression or whether it can be explained by artifacts such as different styles of responding to questions, and differences in openness. These examinations have concluded that the gender difference appears to be a true difference in the experience of depression (Gove & Tudor, 1973; Nolen-Hoeksema, 1987; Weissman & Klerman, 1977).

Men and women have different response styles in which men distract themselves whereas women ruminate on their depressed mood, and therefore amplify it (Nolen-Hoeksema, 1987). One study of explanatory style (i.e., one's style of generating explanations for causes of positive and negative events) and depression in childhood (Nolen-Hoeksema, Girgus, & Seligman, 1991) found that boys reported *more* depressed mood than girls as well as a more ruminating, less effective explanatory style compared to girls. Gender role socialization in early adolescence, related to the biological changes of puberty that heighten an identity with one's gender, is thought to produce the observed reversal of these gender differences by mid-adolescence.

Another explanation for increased experience of depression among girls is evidence that girls experience more challenges and stressors in early adolescence (Petersen et al., 1991a; Simmons & Blyth, 1987). For example, girls are more likely than

boys to go through puberty before or during the transition to secondary school. In addition, several studies have reported that parental divorce is more likely for girls than boys in early adolescence (e.g., Block, Block, & Gjerde, 1986; Petersen, Sarigiani, & Kennedy, 1991b). Pressure on girls to be popular and attractive may also represent an increased challenge to them; related to this factor may be increased pressure on girls to have sex or to at least compete in a responsive way for the attention of boys. These pressures appear to be particularly acute for early maturing girls (e.g., Stattin & Magnusson, 1990). Early maturing girls, especially if they lack the protection afforded by parental supervision, are susceptible to the attentions of older adolescent boys or even young men who may see easy sex in physically well-developed but socially unsophisticated young adolescent girls. At some point, these girls recognize that they have been used and feel depressed about their inability to control the situation, an effect compounded if they also have a child to raise because of early, unprotected sex. Even among upper-middle-class adolescents with more parental supervision and protection, those who had experienced unwanted sexual initiation were much more likely to be depressed as young adults (Stemmler, 1992). Sexual abuse may be a major factor in higher rates of depression among girls and women, given the convergence of rates of early sexual abuse around 20-25% (e.g., Bayatpour, Wells, & Holford, 1992; Russell, 1983) and the likelihood of depression as one outcome (e.g., Browne & Finkelhor, 1986).

Both less effective coping styles and more challenges may increase the likelihood of depression among girls. Studies are needed that test this and other hypotheses for the increased emergence of depression among girls compared to boys.

Other risk groups. Rates of depression and depressed mood may be higher among adolescents in some ethnic groups or other subgroups. For example, in a review of community studies of adolescent depression, Fleming and Offord (1990) reported that in two of five studies where race was examined, black adolescents had higher rates than whites. On the other hand, Nettles and Pleck (in press) reviewed several studies and concluded that although African-American youth are a greater risk for many negative behavioral and health outcomes, rates of depressive symptoms are typically lower than in Caucasian youth. In a study of one of the largest multiethnic samples of adolescents, Dornbusch, Mont-Reynand, Ritter, Chen, and Steinberg (1991) reported that Caucasian and Asian-American youth reported more depressive symptoms than African-American or Hispanic-American adolescents, even after controlling for levels of stressful life events. Given other findings (e.g., Fitzpatrick, Fujii, Shragg, Rice, Morgan, & Felice, 1990), it is probably wise to note Hammen's (1991) conclusion that there is no evidence for black-white differences in depression among adults. Rates among Native American adolescents appear to be elevated (Beiser & Attneave, 1982; May, 1983); high rates have been reported especially among Native Americans in boarding schools (Kleinfeld & Bloom, 1977; Kursh, Bjork, Sindell, & Nellie, 1966; Manson, Ackerson, Dick, Baron, & Fleming, 1990).

Further, adolescents living in rural areas may be at a greater risk for depression compared to those in urban or suburban areas (Sarigiani, Wilson, Petersen, & Vicary, 1990; Petersen, 1991; Petersen, Bingham, Stemmler, & Crockett, 1991, July), although Hammen (1991) concludes that there are no effects of living in a city or rural area among

adults. Another group at risk for increased suicide and probably depression may be gay and lesbian youth, with a two- to three-fold risk of suicide (Gibson, 1989). Most of these studies of special groups fail to control for income level or other circumstances, thus perhaps confounding the results. Much more research is needed to identify whether some groups are at greater risk than others. Such studies would also help to pinpoint the developmental processes involved.

Historical trends. The National Institute of Mental Health Epidemiological Catchment Area studies suggest historical increases in depression (Weissman, Leaf, Holzer, Myers, Tischler, 1984). Rates of depression have increased significantly since World War II (Klerman, 1988). Such historical changes may be especially strong with adolescents, and may be associated with similar increases in depression, suicide, and related problems. Although it has been speculated that these increases were effects due only to the size and competition for resources among the "baby boom" cohort (Klerman, 1988), recent cohorts continue to show higher rates of most of these problems, suggesting that increases are not simply due to a larger cohort of youth (Gans, Blyth, Elsby, & Gaveras, 1990).

Summary. Although much more work is needed on the epidemiology of depression in adolescents, existing evidence suggests increased risk of depression in recent decades. Girls and other groups--such as Native Americans and homosexual youth--may have increased risk of depression but too few studies have considered subgroup variations other than gender to permit inferences at this point.

Related Disorders and Problems

One of the most well documented facts about depressive mood, syndromes, and disorders is that they often co-occur with other symptoms and disorders (Compas & Hammen, in press). Co-occurrence among disorders is called comorbidity. When we refer to problems without a clinical diagnosis, problems that tend to occur together are usually said to covary. In examining the comorbidity of disorders and the co-occurrence of symptoms and syndromes it is essential to distinguish between studies with community as opposed to clinical samples. Estimates of true comorbidity and co-occurrence can be obtained only from community samples, as clinical groups will be affected by a number of sources of bias (Caron & Rutter, 1991).

In a large community-based sample, 42% of adolescents who had experienced a depressive disorder had a comorbid disorder, a rate that was significantly higher than expected from the base rates of the disorders and higher than the rate for adults with depression (Rohde, Lewinsohn, & Seeley, 1991). The co-occurrence of disorders was similar for boys and girls, except that boys were more likely to have both disruptive disorders and depression, whereas girls were likely to have eating disorders and depression. Comorbidity was found primarily for substance abuse in adulthood, whereas depression was associated with a larger number of disorders in adolescence.

A wide range of disorders show high rates of comorbidity with depressive disorders in adolescence. For example, the comorbidity of anxiety disorders is estimated at 30% to 70% (Kovacs, 1990). The overlap between depression and conduct disorders is also high, estimated at 20% to 35% in children and adolescents (Kovacs et al., 1984; Kovacs,

Paulauskas, Gatsonis, & Richards, 1988). Further, a high incidence of personality disorders has been reported among depressed adolescent patients (Clarkin, Friedman, Hurt, Corn, & Aronoff, 1984; Yanchyshyn, Kutcher, & Cohen, 1986).

A high proportion of suicide attempters are depressed, at least after the attempt (Rotheram-Borus & Troutman, 1988; Spirito, Overholser, Ashworth, Morgan, & Benedict-Drew, 1988). Depressed mood appears to be a strong predictor of suicidal ideation (or thoughts) (Kandel, Raveis, & Davies, 1991; Lester & Miller, 1990). Although 83% of youth with suicidal ideation show signs of depression (Carlson & Cantwell, 1982), the ratio of depressed to depressed suicidal adolescent males has been estimated as 660:1 (Shaffer & Bacon, 1989). Depressed mood, drug use, and suicidal ideation are strongly related (Block & Gjerde, 1990; Kandel et al., 1991; Levy & Deykin, 1989). Conduct disorders (and especially impulsivity), depression, and substance use disorders together predict frequency and lethality of suicide attempts (Frances & Blumenthal, 1989). Further examination is needed of the causal processes linking substance use, impulsivity, depression, and suicide.

Eating disorders and substance abuse also frequently co-occur with clinical depression (Attie, Brooks-Gunn & Petersen, 1990; Katon, Kleinman, & Rosen, 1982; Rivinus et al., 1984). Similarly, extreme weight and eating concerns also covary with depressed mood (Post & Crowther, 1985; Richards, Boxer, Petersen, & Albrecht, 1990; Rosen, Gross, & Vara, 1987). At least in girls, poor body image may lead to eating disorders, and then to depression. Again, however, the developmental sequence needs further clarification.

Several studies show elevated depression with medical illness (Cavanaugh, 1986; Fitzpatrick, Barry, & Garvey, 1986). This relationship is usually interpreted as stress and anxiety of the medical illness causing the depression. It also seems possible that depression may make one vulnerable to medical illness through disruption in health related behaviors and/or through common underlying biological pathways in depression and some forms of physical illness.

Depression may cause other problems through its effects on interpersonal functioning (Hammen, 1991). Deficits in interpersonal functioning among depressed individuals are thought to produce poor relationships between parent and child as well as with romantic partners. This hypothesis seeks to explain the three-fold elevation of pregnancy among school-aged girls with depressive symptoms (Horwitz, Klerman, Sungkuo, & Jekel, 1991). These girls tended to have poor relationships both with their mothers and with their partners; their deficits in interpersonal functioning may have limited their capacity to negotiate a sexual relationship and may also have left them vulnerable to feelings of loneliness, increasing the attractiveness of having a baby to love. As discussed earlier, a history of sexual abuse may be the underlying factor in this pattern.

In summary, depression and depressed mood are related to a broad spectrum of other disorders and problems in adolescence. Whether depression causes these problems or the other disorders, or whether other problems cause depression remains to be clarified. There is some evidence for both directions of causation.

Developmental Processes: Risk and Protective Factors

Reviews of research document the challenges of adolescence (e.g., Feldman & Elliott, 1990; Petersen, 1988a), a phase of life characterized by change in every aspect of individual development as well as in every major social context (e.g., Petersen et al., 1991a). The biological changes of puberty as well as the social changes related to the move from elementary to middle or junior high school may be considered primary, with other changes derived from one or both of these. For example, puberty affects body and self image as well as how the adolescent is seen by others. Similarly, the move to a larger secondary school affects the peer group and friendships.

Like other phases of the life course, adolescence also includes experience of stressful life events, with some likely to be more frequent or stressful at this age (Camarena, Sarigiani, & Petersen, 1990). For example, school changes are more frequent in adolescence and parental divorce may have stronger impact on some aspects of development at this age (e.g., romantic relationships). Thus, the extent of potentially difficult changes in adolescence predicts increased psychological difficulty. Depression stands out among the psychological problems of adolescence, both for its significance for adjustment during the adolescent years and its long-term effects on psychological functioning in adulthood.

Biological Processes

Among adults, there is evidence that biological dysregulation occurs with depressive episodes (Akiskal & McKinney, 1973; Shelton, Hollon, Purdon, & Loosen, 1991). The search for clear biological markers of depression in adolescence is in its early

stages, and the findings to date have been inconclusive. For example, unlike adults who tend to secrete more cortisol when depressed (Asnis et al., 1985; Sachar, Puig-Antich & Ryan, 1985), some studies have found that depressed children and adolescents tend to secrete less cortisol (Dahl, Ryan, Puig-Antich, Nguyen, al-Shabbout, Meyer, & Perel, 1991; Kutcher & Marton, 1989). On the other hand, it appears that the sleep onset mechanism is impaired in some depressed adolescents, as evidenced by decreasing growth hormone secretion, increasing cortisol, and increasing the time until sleep in suicidal depressed adolescents (e.g., Dahl, Ryan, Williamson, Ambrosini, Rabinovich, Novacenko, Nelson, & Puig-Antich, 1992). Further attention needs to be given to differences among subgroups of depressed adolescents and to the timing of biological measures (e.g., sleep onset vs. 24-hour averages).

Genetic factors are also implicated with depression. Affective disorders tend to run in families (Andreasen, Endicott, Spitzer, & Winokur, 1977; Gershon et al., 1982; Weissman et al., 1984). Identical twins are four to five times more likely than fraternal twins to show concordance for major depression (Kendler, Heath, Martin, & Eaves, 1986; Wender et al., 1986). The genetic loading for childhood and adolescent depression may be higher than that for depression whose onset occurs in adulthood, although additional research is needed to examine genetic and environmental contributions (Puig-Antich, 1987; Strober et al., 1988). It also appears that earlier onset of depression is predictive of more frequent and severe depressive episodes (Kovacs et al., 1984a; Strober, 1983). Most family studies have failed, however, to disentangle genetic and environmental processes that may predispose offspring of depressed parents to depressive outcomes.

Psychological Factors

As mentioned earlier, negative body image is thought to lead to depression and eating disorders (Attie & Brooks-Gunn, 1991; Post & Crowther, 1985). Low self-esteem may also lead to depression (Harter, 1990; Renouf & Harter, 1990). Anxiety typically precedes depression, suggesting a causal role (Finch et al., 1989; Kovacs, 1990; Suomi, 1991). The psychological processes related to depression may be different for boys and girls; in one study, boys who were depressed at 18 years of age were aggressive, self-aggrandizing, and had poor impulse control in pre-school, whereas depressed 18-year-old girls were overcontrolled and inhibited in pre-school (Block, 1991).

Cognitive Factors

Adolescents show dramatic increases in cognitive ability and reasoning capacity (Graber & Petersen, 1991; Keating, 1990). This increased capacity to reflect on the developing self, and the future, is thought to play a role in the possibility of experiencing depressed mood. Hammen (1990) outlined three general approaches to research on cognitive vulnerability to child/adolescent depression, including information processing models, depressive attributional style, and self-control cognitions. There is evidence of differences between depressed and non-depressed adolescents in each of these aspects of cognition; however, the role of cognitive processes as causal factors in depression is far from clear. For example, children and adolescents who attribute negative events to internal, stable, and general (rather than specific) causes are more likely to be depressed (Kaslow, Rehm, & Siegel, 1984). A recent longitudinal study indicates that attributions may emerge as important correlates or predictors of depressive symptoms in later

childhood and early adolescence but are unrelated to depressive symptoms in childhood (Nolen-Hoeksema, Girgus, & Seligman, 1992). Further longitudinal research is needed to clarify the temporal relations between cognitive processes and depression.

Family Factors

Having a depressed parent is a major risk factor for depression in childhood (see reviews by Downey & Coyne, 1990; Hammen, 1991). Offspring of depressed parents are more likely than children of healthy parents to experience perinatal complications, cognitive impairments in infancy, school problems, peer problems, and high rates of depressive disorders as well as other psychiatric disorders and problem behaviors. Both genetic and psychosocial processes are likely to be involved with these outcomes.

Multiple mechanisms appear to be involved in the transmission of depressive disorders from parents to their children. These include genetic predisposition (Weissman, 1990), emotional unavailability of parents (Lee & Gotlib, 1991), dysfunctional parent-child interactions (Burge & Hammen, 1991), and marital conflict (Downey & Coyne, 1990). Low family cohesion and expressiveness (e.g., Freidrich, Reams, & Jacobs, 1982; Reinherz, Stewart-Berghaur, Pakiz, Frost, & Moeykens, 1989) as well as family conflict (e.g., Carlton-Ford, Paikoff, & Brooks-Gunn, 1991) are associated with depressive symptoms in children. Parental divorce also appears to amplify behavioral disturbances and depression in adolescents (Block et al., 1986; Cherlin et al., 1991; Sarigiani, 1990). Marital discord and economic hardship lead to higher incidence of depression in adolescents (Asarmov & Horton, 1990; Lempers & Clark-Lempers, 1990).

Peers

Low peer popularity is related to depression and depressive symptoms (Jacobsen, Lahey, & Strauss, 1983). Among young adolescents in one study, less closeness with a best friend, less contact with friends, and more rejection experience contributed to increases over time in depressive affect (Vernberg, 1990). Conversely, being depressed appears to contribute to poor relationships. There is some evidence that poor peer relationships constitute a risk factor for depression in early adolescence, but good peer relationships at this age do not appear to provide a protective influence. Later in adolescence, close peer relationships do appear to be protective, particularly when parent relationships are impaired in some way (Petersen et al., 1991b; Sarigiani, 1990). Poor peer relationships in adolescence are among the strongest predictors of adult disorder (Sroufe & Rutter, 1984).

School Factors

The course grades of both boys and girls appear to decrease over adolescence (Schulenberg, Asp, & Petersen, 1984; Simmons & Blyth, 1987). Although some part of this decline is due to increasingly difficult grading practices as students move from elementary to secondary school, depression also may play a role. In one study (Ebata & Petersen, 1992), boys who were depressed and who engaged in minor delinquent activities had sharp grade declines relative to those who were only depressed or only delinquent. Boys with no depressive episodes and no delinquent activities showed stable achievement over the course of adolescence. Interestingly, there was no relationship between depression and achievement for girls.

Daily Stress and Stressful Life Events

The experience of difficult changes or challenges in adolescence appears to predict increased depressed affect. Parental divorce is one such change whose effects were described earlier. As another example, both boys and girls who went through puberty before or at the same time as a move from elementary to secondary school reported more depressed affect than adolescents who went through puberty after their school transition; interestingly, the effects of becoming pubertal prior to changing schools increased over the course of adolescence so that the differences in depressed affect were more pronounced by the end of high school (Petersen et al., 1991b).

Studies show, however, that daily stressors mediate the association between major stressful events and psychological symptoms (Wagner, Compas, & Howell, 1988). Major life events appear to exert their effects on psychological well-being by creating daily stress, and by weakening resources (Compas, Howell, Phares, Williams, & Ledoux 1989). Depressed adolescents report more acute and chronic stressors than youth with conduct disorder, with rheumatic disease, or healthy youth with a number of life stressors; in addition, depressed adolescents report fewer social resources (Daniels & Moos, 1990).

Developmental Trajectories

At present, several developmental pathways appear plausible for depression during adolescence. For example, environmental events may trigger biological dysregulation through dysregulation in the social fabric of an individual's life (Ehlers, Frank, & Kupfer, 1988). Alternatively, depression might result from a series of events and processes, including genetic susceptibility, biological insults, temperament and other individual

characteristics, environmental events, developmental changes, and coping responses available to the individual. Biological changes might serve to heighten and maintain the psychological distress experienced by a depressed individual (Whybrow, Akiskal, & McKinney, 1984). A third causal pathway for depression might be chronic or extreme stresses that become difficult and overwhelming for the individual. The death of a loved one or growing up with a depressed parent both constitute strong risk factors for depression. Some dysfunctional processes involve the perpetuation of interpersonal deficits and depression across generations (Hammen, 1991).

Once on a depressed trajectory in development, an individual appears more likely to stay on this course because of the tendency to withdraw from the very social supports that can minimize negative effects. These effects are likely to be especially devastating to a developing adolescent. Imagine a thirteen-year-old, hospitalized for depression following the death of a parent. The hospitalization removes the adolescent from the peer group and school; family members are likely to visit, but the context is certainly not the same as home. This adolescent not only experiences unusual and perhaps stigmatizing treatment, but is likely to miss important developmental experiences, especially at school and with peers. The return to these contexts after hospitalization is likely to be very difficult. The divergence from the normal developmental pathway in adolescence during treatment for depression may cause, or perhaps reinforce, a more isolated, depressed approach to life. Research is needed that examines the effects of treatment, and especially ways to minimize possible negative effects. Further, the identification of developmentally supportive treatments is needed.

Good relationships with parents, and by mid-adolescence with peers, appear to buffer negative effects of stressful life events. Children of depressed parents may not experience these protective relationships, thus exacerbating any genetic vulnerability that may exist. Again, treatment of or intervention with these children is crucial.

Approaches to Intervention: Treatment and Prevention of Adolescent Depression

Treatment of Adolescent Depression

Pharmacotherapy. Building on research with adults, several varieties of psychoactive medications have been evaluated as treatments for adolescent depression. The characteristics and results of both open trial (uncontrolled) and placebo controlled studies on the efficacy of pharmacotherapy for adolescent depression are summarized in Table 6. The results of these studies fail to support the efficacy of pharmacotherapy for adolescent depression. That is, in the only two double blind placebo controlled studies the treatment and placebo groups did not differ significantly after treatment. Further, there are reports in the clinical literature of sudden and unexplained deaths of children and adolescents who were treated pharmacologically for depression (Geller, 1991; Riddle et al., 1991). Given the efficacy of these treatments with adults, researchers have speculated on the reasons for the lack of results and perhaps negative effects. The possibilities range from the different biological substrate of adolescents to the possibility that adolescent depressives have more serious forms of depressive disorders because of earlier onset and high rates of comorbid disorders such as anxiety.

Psychosocial and psychotherapeutic treatments. Psychosocial and psychotherapeutic interventions in the treatment of adolescent depression include cognitive-behavioral therapy (Lewinsohn, Clarke, Hops, & Andrews, 1990), psychodynamically-oriented therapy (Bemporad, 1988), family therapy (Lantz, 1986), social skills training (Fine, Forth, Gilbert, & Haley, 1991), and supportive group therapy (Fine et al., 1991). Table 7 compares the effectiveness of various forms of psychotherapeutic and psychosocial interventions with other interventions or wait-list controls. The three studies that included no treatment control groups all provide some confirmation that depressed mood or, in one case, depressive disorders, can be significantly reduced through treatment (Kahn, Kehle, Jenson, & Clark, 1990; Lewinsohn et al., 1990; Reynolds & Coats, 1986). Improvement, with large effects, was found on self-report measures of depressive symptoms. There was also some evidence that these gains in reductions in depression were maintained over longer periods of time. Surprisingly, no evidence has been found for the superiority of any one form of psychological treatment over another. These studies also suggest that treatment effects are not limited to depressive symptoms; significant reductions have also been found in anxiety symptoms (Lewinsohn et al., 1990; Reynolds & Coats, 1986).

No research has yet integrated the effects of pharmacotherapy and psychotherapy for depressed adolescents. Attention to individual differences in response to treatments as well as to the overall effectiveness rates of these interventions appears to be a more productive avenue for research. It would be useful to know whether there are subgroups who show different responsiveness to pharmacologic versus psychosocial treatment.

Prevention of Adolescent Depression

Based on the high rates of depressed mood, depressive syndromes, and depressive disorders that occur during adolescence, it is clear that treatment efforts will never be sufficient to meet the full needs of the population. Professional resources are inadequate to meet these needs, both in terms of the limited number of trained, available professionals and in terms of barriers to access to treatment that are confronted by adolescents, especially adolescents living in poverty. Therefore, prevention of the spectrum of adolescent depressive problems is of paramount importance if the needs of the largest number of adolescents are to be met.

Prevention of adolescent depression can take two forms. First, preventive services can be delivered to entire populations of adolescents based on the assumption that all adolescents are at some risk for experiencing at least depressed mood, if not for the development of depressive disorders. The assumption behind such programs is that all adolescents are exposed to sources of risk for depression, such as stressful life experiences, and that enhancing their ability to respond adaptively to these risk factors will reduce the incidence of depression in the population. This approach further assumes that depressive symptoms exist on a continuum and that subclinical levels of depressed mood or negative affect may serve as markers of increased risk for the development of depressive disorders. By reducing depressed mood in the population, the overall risk for depressive disorders may also be reduced. Second, adolescents who have been exposed to some identifiable risk factor can be selectively targeted for preventive efforts. Most importantly, adolescents whose parents are clinically depressed are known to be

at increased risk for depressive disorders as well as other problems compared to the population at large. Services can be delivered selectively to those at greatest risk and in greatest need.

Population-wide prevention programs. In spite of the promise that broad-based preventive interventions could contribute to reduction in the incidence of depression, no controlled (i.e., with appropriate comparison samples) evaluation studies of such programs have been published. The ongoing research of Petersen and colleagues (1988b) promises to provide the first such data concerning the effects of a preventive intervention for young adolescents.

Broadly focused programs designed to promote social competence and problem solving skills also have relevance for the prevention of depressive mood and symptoms at the population level (Weissberg, Caplan, & Sivo, 1989). This general family of interventions has been designed to teach the social competencies and life skills needed by adolescents to foster positive social, emotional, and academic development. Although problem-solving interventions have been found to influence a wide variety of outcomes, measures of depressed mood, syndromes, or disorders have not been included in evaluations of these programs. Assessment of depression will be an important criterion to include in future evaluations of social competence promotion programs.

Although there are few prevention programs targeting depression specifically, a number of preventive interventions have been developed to address stressors and problems that may be linked to depression. For example, several programs have been developed to assist adolescents in coping with the stress of divorce (see Grych &

Table 6
Evaluation Studies of Pharmacologic Intervention for Adolescent Depression

Study	N	Sex	Age	Diagnosis ¹	Treatment	Design	Controls	Results ²
Dugas et al. (1985)	80	40 male 40 female	8-19 years	None	Mianserin	Open fixed dose	None	Significant effects on each of four separate criteria
Ryan et al. (1986)	34	17 male 17 female	10-17 years	MDD, RDC criteria	Imipramine	Open fixed dose	None	44% improved; MDD only responded better than MD and Anxiety Disorder
Ryan et al. (1988)	23	11 male 12 female	11-18 years	MDD, RDC criteria	MAOI with or without TCAs	Open	None	74% fair or good clinical response regardless of dietary compliance 57% fair or good clinical response with dietary compliance
Geller et al. (1990)	52	25 male 27 female	12-17 years	MDD, RDC and DSM-III criteria	Nortriptyline	Fixed dose	Double blind	Only 1 active responder of 12 who completed treatment
Strober et al. (1990)	35	11 male 24 female	13-18 years	MDD, RDC and DSM-III criteria	Imipramine	Open fixed dose	None	30% improved
Boulos et al. (1991)	30	Both males and females Number not specified	15-20 years	MDD, DSM- III criteria	Desipramine	Fixed dose Placebo controlled	Double blind Placebo group Placebo washout phase	50% (n = 15) of medication group and 33% (n = 10) of placebo shared improvement

1. MDD=Major Depressive Disorder; RDC=Research Diagnostic Criteria

2. MD=Mood Disorder

Table 7

Evaluation Studies of Psychological Interventions for Adolescent Depression

Study	N	Sex	Age	Diagnosis ¹	Treatment ²	Design	Controls	Results
Reynolds & Coats (1986)	30	11 male 19 female	M=15.7 years	None	CBT Relaxation	Random	Wait-list	Significant decreases on self-reports of depressive symptoms for both CBT and relaxation at post-treatment and 5-week follow-up Relaxation group reported less anxiety than controls but did not differ from CBT post-treatment
Kahn et al. (1990)	68	33 male 35 female	10-14 years	None	CBT Relaxation Self-Modeling	Random	Wait-list	All treatments showed improvement on self-reports of depressive symptoms relative to controls
Lewinsohn et. al. (1990)	59	28 male 31 female	M=16.1 years	MDD DSM-III and RDC criteria for depression	Adolescent CBT Adolescent and parent - CBT	Random	Wait-list	Significant reductions for both treatment groups relative to controls in diagnoses of depression and in self-reports of depressive and anxious symptoms
Fine et. al. (1991)	66	11 male 55 female	M=15 years	MDD and Dysthymia DSM-III-R criteria	Support Group Social Skills Group	Quasi- random	None	Equal gains on self-reports of symptoms for both treatment groups at 9 month follow up

1. MDD=Major Depressive Disorder; RDC=Research Diagnostic Criteria

2. CBT=Cognitive Behavior Therapy

Fincham, 1992, for a review). Although the effects of these programs have been reported with regard to anxiety symptoms and other internalizing problems, measures of depressed affect or symptoms have not been included.

Prevention programs for adolescents at risk for depression. Although population-wide interventions may provide some level of protection against sources of risk for depression for many adolescents, others may be exposed to conditions and circumstances that present a more profound risk for the development of depressive syndromes and disorders. The basic skills that can be taught in broadly-focused programs described above may not be sufficient for individuals faced with heightened risk for depression. Secondary preventive services can be directed to those groups who are identified on the basis of a marker of their increased risk.

Compelling evidence has been summarized above indicating that the single most powerful source of risk for adolescent depression is the presence of a depressive disorder in a parent (Downey & Coyne, 1990; Fendrich, Warner, & Weissman, 1990; Phares & Compas, 1992). The diagnosis of depression in parents serves as a distinct marker of the need for either a preventive or treatment-oriented intervention for the offspring of these individuals. It would not be an overstatement to say that the need for services for children of depressed parents as closely approximates a *prescriptive* recommendation as can be found in the mental health professions. Further, the incidence of major depressive disorders in offspring of depressed parents increases substantially during adolescence, indicating that young adolescents whose parents suffer from depression are a group in particularly high need of services (Beardslee, 1990). Initial

steps have been taken in the development of preventive interventions for depressed parents and their families by Beardslee and colleagues (Beardslee, 1990; Beardslee et al., 1992; Beardslee & Podorefsky, 1988). The intervention is guided by research on the characteristics of adolescents who have displayed resilience in the face of depressive disorders in their parents.

Integrating Treatment and Prevention Efforts

Unfortunately, the resources to support mental health services in the United States are limited and fall far short of the economic and human resources needed to address mental health problems. It is even more unfortunate that limits on these resources have contributed to conflict among mental health professionals regarding the best way to expend these limited resources. In particular, battle lines have been drawn by proponents of prevention and those who favor treatment (Albee, 1982).

With regard to adolescent depression, the battle between proponents of prevention and treatment is one that we cannot afford to fight. Services aimed at alleviating the pain and misfortune associated with depression during adolescence must reflect a broad effort to provide coordinated prevention and treatment programs. This position is based on the assumption that no one approach to providing services for adolescent depression will be sufficient to address the problem. While we fully expect that primary prevention programs will be able to reduce levels of depressed mood and deter the development of more severe forms of depressive syndromes and disorders, they will not provide a sufficient dose to interrupt the development of depression in a substantial portion of the population. The addition of secondary prevention programs aimed at high risk groups should provide

a second net to catch and help many adolescents who, because of exposure to high risk circumstances such as parental depression, conflict, or divorce, were not helped by primary preventive efforts. Finally, treatment programs will remain a necessity because many adolescents who are exposed to conditions of profound risk will not be able to gain access to secondary prevention services to prevent the onset of more serious depressive disorders. Moreover, the majority of adolescents may have insufficient access to treatment services (Keller, Lavori, Beardslee, Wunder, & Ryan, 1991).

Primary prevention programs at the population level, secondary prevention services for high risk groups, and treatment for adolescents who manifest severe forms of depressive disorders must be delivered in a coordinated, sequential fashion. Evidence is accumulating to indicate that interventions at all three of these levels need to address dysfunctional cognitive processes, skills for coping with acute and chronic stress, and strategies to deal with interpersonal relationships and problems. If these themes are addressed in primary and secondary prevention programs as well as in treatment, they would contribute to the development of a core set of competencies for all adolescents. The foundation for these skills would be laid in a preventive intervention delivered at the population level (Petersen, 1988b). Further development of these competencies could then be pursued in secondary prevention programs for adolescents whose parents are suffering from clinical depression or other sources of risk (Beardslee, 1990). Finally, for those adolescents who warrant further intervention, these same skills would continue to be the target in psychotherapeutic interventions (Lewinsohn et al., 1990). Continuity of

this type across interventions could enhance the effects of our efforts to alleviate adolescent depression at each step in the developmental process.

Implications for Research, Program, and National Policy

Implications for Research Policy

Several inferences follow from the nature of the research that we discovered in our review. First, most of the studies classified under "adolescent depression" are based on clinical populations and use diagnostic measures. Second, a high percentage of studies published since 1987 focus on children of depressed parents. Third, most of these studies consider depression as an outcome of other conditions or influences while only a few examine depression as a cause, for example, of suicide or poor school achievement. Fourth, we could find few studies that examined protective factors; of note, individual factors were more likely to be considered only as risk factors whereas social influences provided most of the instances of protective factors (but social influences were more likely to be construed as risky than protective). Finally, the current literature provides little basis for differentiating processes leading to depressive disorder, depressed mood, self-esteem, and other problems such as drug abuse or conduct disorders.

Although the large quantity of existing research on adolescent depression may suggest that no additional funding is needed to stimulate work in this area, past research has been concentrated in only some of the key areas. Most notably lacking are studies that integrate biological and social processes in the etiology of depression during adolescence. (And, as many have argued, understanding the etiology of depression at

adolescence would be tantamount to understanding the etiology of depression.) Also lacking are studies that examine whether there are different causes of clinical depression, of depressed mood, and of milder unhappy states such as poor self-esteem or general unhappiness. Both the biological studies and the familial studies of clinical depression have identified information that differentiates bipolar from unipolar outcomes, but they have not extended this work to clinical samples. Conversely, studies of psychosocial processes tend to use non-clinical samples and lack diagnostic methods. As this review demonstrates, sufficient research exists to formulate clear hypotheses that begin to clarify how depression, in all three forms, develops.

Similar observations result from current intervention research. Although the studies of biological treatments rigorously pursue the specific neurobiological processes involved, nonbiological treatment studies are less clearly based in theories about process. Intervention studies could illuminate understanding of etiology if well conceptualized and designed.

Having depressed parents is the most consistent risk factor for adolescent depression. Although growing up with a depressed parent presents an enormous risk for the development of depression and other disorders in offspring, not all children of depressed parents display dysfunctional patterns of development. Therefore, comparison between siblings within families of depressed parents is an important avenue for future research. Comparisons among siblings whose parents are depressed may offer clues to the non-shared characteristics of family environments and genetic factors which either increase the risk for disorder or protect children from disorder in these families. Further,

data of this type should be useful in constructing interventions for families with depressed parents, as they could provide information about both risk and protective factors for adolescent depression.

Implications for Program Policy

A major implication emerging clearly from the existing research on adolescent depression is that children in families with depressed parents are at significant risk for developing depression and other psychopathological problems. Treatment programs for depressed parents must consider treatment for their children. Without such an approach, programs treating adult depression are only treating part of a problematic family dynamic and ignore the significant effects on offspring.

The possibility that pharmacological treatments may work differently with adolescents than adults has significantly illuminated the nature of the processes involved across the life course in depression. It is important that information about the differences in pharmacological treatment of adolescent versus adult depression be communicated to clinicians. Specifically, clinicians should exercise caution in prescribing pharmacological treatments for depression in adolescence.

If, like diabetes, depression is triggered by situational factors and exacerbated by biological vulnerability, significant opportunity exists for multiple levels of preventive intervention. For example, life skills training programs show promise of providing coping skills to enable young people to deal with situations that might precipitate depression. Such programs would be especially important if the challenges of adolescence have

increased relative to earlier historical times. Any efforts that reduce morbidity among youth would surely be important to pursue as a policy matter.

Another major programmatic implication is the comorbidity of disorders and covariation among adolescent problems. These results suggest that categorical and single problem focused interventions are inappropriate. Instead, multivariate, person-focused, comprehensive approaches are likely to be more effective with adolescent depression.

Implications for National Policy

At the national level, consideration should be given to the reorganization of research funding. The United States Congress Office of Technology Assessment (OTA) report (1991) on adolescent health highlighted the low national priority accorded to depression research and treatment. The report documented significant challenges confronting adolescents. The three major options described in the OTA report would also benefit research and programs related to adolescent depression: (1) improve US adolescents' access to health services, (2) restructure and invigorate federal efforts to improve adolescent health, and (3) improve adolescents' environments. Specific strategies are identified in each of these areas. Further, the report provides specific options related to mental health problems. All these approaches would make major contributions to our understanding, prevention, and treatment of adolescent depression.

Adolescent depression is a major, pervasive, and perhaps increasing problem. A sufficient knowledge base now exists to guide action, including prevention and treatment efforts. Significant morbidity of adolescence and adulthood could be ameliorated by attention to adolescent depression.

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